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SURGICAL ANEMIA AND RESUSCITATION.[1]

By George W. Crile, M.D.,
PROFESSOR OF CLINICAL SURGERY IN THE WESTERN RESERVE UNIVERSITY,
CLEVELAND, OHIO.

It so happened that at the time I was invited to deliver this lecture, my research associate, Dr. D. H. Dolley, and I had reached a point in the investigation of one phase of the subject of surgical anemia, at which, in conjunction with my own previous experimental and clinical work, tentative generalizations seemed warranted. The following statements include, for the most part, generalizations of experimental and clinical observations. I have drawn heavily on the histological work of Dr. Dolley. It is not possible on this occasion to refer to the many valuable contributions he has made. From the literature I have drawn liberally upon the work of D'Halluin, Hill, Stewart, and his associates, as well as upon that of Sarbo, Marinesco, and Mott. A detailed publication of the data upon which the generalizations are based is contemplated later.

In the vegetable and in the animal kingdom functional activity and growth require an almost constant supply of food. Impairment or loss of this constant supply is followed by impairment or loss of function or by death. Increased or diminished food supply affects growth and function in infinite ways. Owing to environment and ultimate design, species respond differently to the loss of food. Neither individuals as a whole, nor their various parts, respond equally to a diminished or a complete loss of food. The histological elements of a given organ endure the various degrees of impairment or loss of function differently. Death of an animal, therefore, falls unevenly upon its various tissues and organs. Undoubtedly, postmortem examinations and burials are made while some of the tissues of the body are still living. The vital spark may smoulder or it may be extinguished. Death may be definitive or relative. Definitive death is a vital and final resolution and is irremediable. Relative death is a state of suspended animation.

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[1] The Mütter Lecture of the College of Physicians of Philadelphia, delivered December 11, 1908.

Suspended animation is susceptible of re-animation or resuscitation. Suspended animation, as understood for our present purpose, is produced by a loss of food supply, and re-animation or resuscitation may be accomplished only by a supply of the required nourishment. Whether the needed chemical elements are supplied in gaseous form as oxygen, or in chemical combination as proteids, they all constitute food. The loss of this complex food supply will, for our present purpose, be designated anemia, and the anemia most frequently met in surgical practice I will designate surgical anemia.

Surgical anemia may be due to absolute loss of blood volume (hemorrhage); to segregation of the blood in the vascular system by disturbance of the vascular mechanism, as in shock or collapse; to local interruption of the circulation by mechanical means, such as thrombosis or embolism, strangulations, torsions, tourniquet, bandages, apparatus, pressure, etc. Whether considered with reference to the entire body or to a part, the result of a given anemia, whether partial or complete, is dependent upon the susceptibility or the resistance of the affected part to anemia.

It is not my purpose at this time to consider hemorrhage, shock, collapse, and the various mechanical agencies alluded to, any further than as to their role as a cause of surgical anemia. Disregarding, then, the mechanism of the cause of anemia, I will in a general way, as they relate to the practice of surgery, consider the phenomena of anemia and its treatment, namely, resuscitation.

To approach this subject, we first need to know the relative endurance of the various tissues and organs to anemia, hence their resuscitability. Turning to this point of our inquiry, we are at once confronted by many difficulties, the principal being the various degrees of susceptibility of not only organs as a whole, as the kidney and the brain, but the individual difference in susceptibility of the component cells of the various organs. Furthermore, certain functions of the organ may be lost while the cells performing these functions may survive. Again, some functions of the cells may be retained, while others may be lost. Finally the loss of function may be temporary; if so, have these cells been partially reconstructed physically? Many of these questions we are not prepared to answer. Indeed, as they relate to the practice of surgery many are not important. The finer metabolic changes, the ultimate method of re-animation or regeneration, may be left to the trained physiologist to determine. What we, as practitioners, want to know is, how long may the various organs and tissues be deprived of their circulation and still be susceptible to resuscitation? How may resuscitation be best accomplished?

The skin may be removed, preserved for at least several weeks, then on proper planting may grow again. The voluntary muscles enter into a state of rigor mortis after an anemia of about twelve hours. The nerves ending in the muscles degenerate after a shorter period. The bones endure an anemia longer than the muscles. The tendons and connective tissue occupy a position probably between muscle and bone. In a decubitus, as a paralytic bedsore, one observes early necrosis of all the tissues lying between the skin and the bone, showing their greater susceptibility. As to the viscera, the kidney may endure anemia and anatomically survive after a total anemia of at least one hour. The intestinal tract may anatomically and physiologically survive between six and seven hours. The heart (Kuliabko) has been isolated and kept in refrigeration for forty-four hours and on perfusion with oxygenated defibrinated blood has again beat rhythmically. Bloodvessels have been transplanted after isolation for many weeks and have been able to perform at least their passive function (Carrel, Guthrie). Just how long they may retain their viability has not been precisely determined.

The several parts of the central nervous system endure anemia in varying degrees. The respiratory centre may endure an anemia of from thirty to fifty minutes; the vasomotor and cardiac centres, about twenty to thirty minutes; the spinal cord, eight or ten minutes; the motor cortex, eight or ten minutes; that part of the brain presiding over consciousness, the intellect and the psychic state, six to seven minutes.

The foregoing are approximate figures and include only the grosser functions. They permit certain generalizations: (1) Tissues and organs of low specialization endure total anemia better than tissues and organs of high specialization; (2) in the central nervous system the component parts endure anemia according to their phylogeny (Stewart). The nerve tissue presiding over the basal functions of life endures anemia better than the nerve tissue which by the process of evolution differentiates the higher animals from the lower; (3) the weakest link in the entire vital chain in the central nervous system is that which presides over conscious life and its special manifestations; (4) the higher the development of the tissue the more sharply is the period of endurance to anemia marked. While the vasomotor and the respiratory centres may show very considerable variation in their resistance to anemia, the cerebrum varies but slightly.

The histological changes will be alluded to later. Having surveyed in a general and summarizing way the susceptibility to complete anemia of the various tissues and organs that may be of surgical interest, the natural corollary is an inquiry into the methods of resuscitation.

Resuscitation may be considered with reference to organs or tissues individually or to the body as a whole. The best resuscitation is prevention. Here, again, as in the inquiry as to the length of time the tissues and organs may endure total anemia, the subject is so large that details must be sacrificed to a survey of the general field. One is tempted to discuss the ischemic palsy of tight splints, forgotten Esmarch bandages, tourniquets, the pernicious use of a bandage applied directly to the skin over fractures, splint contact with bony prominences, making the heel bear the weight of the splint-bound leg, the long unshifted recumbency upon an unprotected glass- or metal-topped operating table, the pressure of the brachial plexus upon the head of the humerus when the hands are firmly tied above the head during operations, the indentations and insufficient protection of plaster-of-Paris bandages, the unchanged posture on unsuitable mattresses of unconscious or paralyzed patients who have lost the beneficent protection of pain, etc. All these ways and means of causing needless deformity, gangrene, and suffering tempt more consideration, but I must hasten to other problems.

In anemia due to external or mechanical agents the cause and the treatment are obvious. When due to occlusion of supplying vessels, is there a newer method of attack? The work of Carrel, Payr, and others has, at least, furnished means of possible substitution. The perfection of the technique of bloodvessel anastomosis, the success of heteroplastic transplantation of bloodvessels, the establishment of the substitution of a vein for an artery, the use of local, intraneural, spinal, and nitrous-oxide-oxygen anesthesia to obviate shock in poor surgical risks, make it possible at least to determine to what extent the cases of thrombosis, embolism, aneurysm, or traumatic solution of continuity of vessel walls may be benefited by repair or by substitution of vessels. In addition, the direct transfusion of blood may meet the resulting emergencies of acute anemia due to shock and hemorrhage.

In the possible transplantation of organs we must reckon with the effect of anemia as well as with the technical difficulties of the operation. In anemia of the somatic and certain visceral parts there is ample warning and fair surgical opportunity; for even if death does occur, compensations, even regenerations, are generous. In anemia of the central nervous system the warning is momentary or absent, the damage wrought is swift and irremediable. Though the results of anemia here are swift and terrible and the resulting loss of function largely without compensation or repair, we still have left to us the vast and useful field of prevention. Among many subjects may be mentioned the prevention of partial but increasing anemia, due to pressure upon the spinal cord following injuries and tumors. In total cross lesion of the cord there remains no direct surgical problem, but in pressure upon the cord, even though there is but the slightest or even doubtful evidence of function remaining, a speedy operative relief should be attempted, lest the resulting increase of pressure due to the consequent swelling or oozing may, by producing complete anemia, extinguish the remaining spark of life. Then, too, one must here reckon with the misleading shock phenomena (Sherrington) immedaitely following the trauma, causing a break in the synapses, mimicking for the time being the picture of a total cross lesion. Hill, Stewart, and others have shown that even a slight "dribble of blood" may for a time preserve vitality.

The brain is frequently subjected to local anemia from a variety of mechanical causes, such as gravity, embolism, ligature, and compression of the common or internal carotid arteries, and increased intracranial pressure.

Embolism of surgical interest may be produced by the washing away of the thrombus formed in the proximal stump of a ligated external carotid. This occurs approximately once in fifty such ligations. Ligation of the common or internal carotids, especially in the state of atheroma, in about one out of three cases, is followed by an anemia so marked that cerebral softening occurs. The younger the subject, the less prone to the sequence of softening. But it is the older and atheromatous subjects that usually require or are supposed to require, ligation. Ligation of the common or of the internal carotid has been most frequently performed as a part of the technique of excision of malignant tumors. A closer study of cancer invasion has shown that cancer attacks the wall of the artery only after it has broken through the capsule of the lymphatic glands and has extensively invaded the deep planes of the neck. It is doubtful whether or not such a case has ever been cured by operation. Our position, then, may be stated as follows: If the cancer has not invaded the wall of the artery, ligation is not necessary; if the wall has been invaded the case offers but a remote if any chance of cure. Is this slight chance worth a 33 per cent. risk of cerebral softening?

In aneurysms the development of the technique is constantly diminishing the number of ligations.

As to the ligation of the common or the internal carotids for the arrest of hemorrhage, our means of successfully combating it by methods not bearing the risk of cerebral softening are almost uniformly adequate. In young subjects the sequence of softening is rarely observed. But what young patient requires such ligation? If, however, ligation must be performed, if time permits, a series of preliminary compressions of the artery, either by applying a clamp directly or by indirect external pressure, apparently widens the collateral blood paths, rendering occlusion less harmful. This is apparently established by both clinical and experimental evidence. Temporary closure of the common carotid by means of a special clamp has been done by me nearly one hundred times. In a few cases I simultaneously closed both common carotids. In addition I have frequently placed the patient in a sharp feet-down inclined posture to enlist the additional aid of gravity in minimizing the venous hemorrhage. The results with respect to the circulation through the brain may be summarized as follows:

In cases of simultaneous closure of both carotids but little anesthetic was required; in one instance respiration failed; in three, recovery of consciousness was delayed. These phenomena were such unmistakable signals of danger from anemia that simultaneous double closure was abandoned. In atheromatous subjects closure of one internal carotid in prolonged operations has been followed by delayed postoperative recovery of consciousness. In one case, in which, in addition, the patient was in a markedly inclined feet-down posture, there was fairly well-marked delirium during three days following the operation. Normal convalescence followed. These were undoubted examples of anemia sequences.

These observations led me further to modify the technique by first closing the common carotid and maintaining the horizontal position until the dissection has been carried to the bifurcation of the carotid, when the clamp is transferred to the external carotid. The blood path now being opened to the brain, no harm follows the inclined feet-down posture. I have observed no anemic phenomena in cases thus handled. Not only does the transfer of the clamp to the external carotid remove the risk of brain anemia, but the closure of the external carotid prevents the moderate bleeding from the reverse current when the common carotid only is closed. Then why is not the external the artery of choice? Because the usual occasion for such a dissection is for the eradication of cancer. Entering the neck to secure the external carotid involves a dissection in a region of frequent metastasis, and so the risk of making a local dissemination of the growth, thus defeating the purpose of the operation. With this exception, the external is the artery of choice, especially in elderly subjects.

After certain operations it is desirable to keep the patient in a sitting or half-sitting posture. In such cases the cerebral circulation, which gravity tends to diminish, may be measurably protected by efficient bandaging over rough elastic cotton to the costal border, or the use of the pneumatic rubber suit under firm distention.

Turning from regional cerebral anemia to the diminution or interruption of the blood current in the supplying arteries, I will consider anemia due to local and mechanical causes originating and operating within the skull. Any consideration of this portion of my theme must represent in a large measure the work of distinguished surgeons of this society.

While the rigid skull gives protection from external violence, every point of such protecting quality becomes a corresponding menace from certain pathological states within. How slight is the factor of safety when the brain is subjected to increased intracranial tension! The principal factor of safety is the displacement of cerebrospinal fluid and the increased general blood pressure in a protective response to the oncoming anemia.

The management of increased intracranial tension may be illustrated by a common form—traumatic hemorrhage. For the purpose of discussing certain pratical points relating to the impaired circulation and consequent anemia, intracrainal hemorrhages may, with respect to their degree, be roughly classified as follows: In the first stage, that of developing stupor, falling pulse rate, and rising blood pressure, the margin of operative risk is ample, complete anemia is not immediately threatened, and the prognosis is good. In the second stage, that of deep stupor, subtotal or total unconsciousness, slow but rhythmic pulse and maximum but even blood pressure, the margin of operative risk is slender, the prognosis uncertain. In such cases all the sails of compensation are set. There is probably only a slight circulation through certain portions of the brain. Even a moderate fall in the blood pressure due to hemorrhage, shock, or anesthetic complications may leave the cortex in total anemia, which if at all prolonged, say seven or eight minutes, will result in fatal degenerations. In the third stage, that of complete unconsciousness, irregular pulse rate and rhythm, high but irregular blood pressure, anemic degenerations may have already occurred, or there may remain a slight dribble of blood forced in at the crest of the oscillating blood pressure waves, perhaps even at the crest of the respiratory wave. This slight circulation may be barely holding the spark of life. Surgical relief by a faultless technique may save an occasional member of this group. In the fourth stage, that in which the pulse is rapid and the blood pressure falling, the powerful blood pressure mechanism has spent its force and is broken, the brain is in complete anemia, and widespread degeneration has occurred. The higher-brain individual is dead, while the remainder of the body lives.

This is merely a crude and artificial sketch of the stages through which the brain may pass in the various degrees of anemia due to increased intracranial pressure. The surgical approach here must be as physiological as possible. Not only must there be no depression of the circulation, but support should be given. The blood pressure should be supported by therapeutic and by mechanical means. The posture should be horizontal or head down, the extremities and the abdomen to the costal borders subjected to pressure or bandaging over cotton. The most important consideration, however, is prompt operative decompression. How many of these cases, still dimly conscious, are subjected to the routine method of "interne" anesthesia, of needless hemorrhage, and a shock producing technique. The diagnosis may be correct, a large mass of clot removed, the circulation and the respiration may at once improve, and the surgeon may be entirely satisfied. All is well, but the patient does not regain consciousness and dies. Such a picture I can vividly recall from personal experience. Causes of increased intracranial tension need strict physiological interpretation.

With this brief and imperfect survey of surgical anemia from local causes, and local resuscitation, I will discuss from the experimental and clinical standpoints resuscitation of the individual as a whole.

After testing current methods and noting their shortcomings, resuscitation was approached from the arterial side, with the following physiological factors as a basis: The physiological researches of Ringer, Kuliabko, Stewart, Sollman, and others had shown that the excised heart could be made to beat again, even many hours after excision, when the coronary arteries were subjected to a considerable pressure from the circulating medium. The researches of Sollman showed that the inauguration of the heart beat was more dependent upon the physical factor of the increased pressure in the coronary arteries than upon the quality of the fluid producing such pressure. He was able to inaugurate cardiac beats by perfusing the coronaries with metallic mercury. The basic problem, then, in resuscitation seemed to us to be that of securing by means of some infusion a coronary pressure in the intact animal to this height by means of cardiac massage alone; it was almost impossible to raise it by means of infusion of artificial sera alone, and generally impossible to raise it to this height by means of cardiac massage and plain infusion combined.

The value of adrenalin in raising the blood pressure, by its action upon the vascular walls in the state of suspended animation, has already been thoroughly established. Introducing the adrenalin into the venous circulation, while easy and practical, had the following disadvantage: the adrenalin first came in contact with the vessels having the least power of influencing the blood pressure, and before a material rise could be affected by its action upon the arteries it was necessary that the solution should pass through the right heart, the lungs, and then back to the left heart on its way to the aorta, then finally affecting the coronary arteries. In a previous research it was found that this too often caused an accumulation of solution and blood in the dilated paralyzed chambers of the heart, defeating resuscitation.

It seemed reasonable to us to suppose that the most direct and effective way of producing a coronary pressure amounting to 40 or more mm. of mercury was by introducing a solution of adrenalin into the arterial system toward the heart. In this way the moment the adrenalin was introduced it caused a contraction of the strong arterial walls, and began to produce an arterial pressure which was communicated directly to the coronary arteries without first passing directly through the already distended and paralyzed chambers of the heart and through the lungs. These considerations were strongly impressed upon us by a clinical case of suspended animation in the course of a cerebellar operation on a child, which was unexpectedly resuscitated by centripetal arterial infusion of adrenalin.

The following will serve as an illustration: An animal was killed by chloroform. A cannula was inserted into the femoral artery and directed toward the heart. After five minutes artificial respirations were begun and the saline solution was allowed to flow for perhaps ten seconds; then a hypodermic injection of from 1 to 2 c.c. of 1 to 1000 solution of adrenalin was given into the tube near the cannula. A few seconds later the blood pressure began to rise steadily; then a few firm pressures upon the thorax over the heart caused a leaping up of high pulse waves, and at the end of three-quarters of a minute the heart beat vigorously, driving the blood up into the infusion bottle, which had been to the height of five feet. The saline injection and the cardiac massage were discontinued, and in a few minutes irregular respirations began slowly, and increased in force and frequency until the normal was established. The animal was then definitely killed.

Having established an effective technique for resuscitation, we then investigated the physiological and histological sequels of the resulting anemia. The laborious task of collecting these data was undertaken by my colleague, Dr. David H. Dolley.

In general the following sequence of return of the various functions and reflexes was exhibited: vasomotor action, respiration, corneal reflex and knee-jerk (tendon reflexes in general), winking, cutaneous reflexes, partial or complete contraction of the pupils, and light reflex. This order was subject to considerable variation, which will be considered under the special discussions of functions and reflexes. Hypertonicity of the voluntary musculature immediately succeeded recovery of a normal tone and was manifested by exaggeration of the knee-jerks, if not by a more or less widespread spastic condition. It always followed rapidly the re-appearance of the knee-jerk. Reflex muscular movements, secondary to skin or tendon stimulation, always preceded those of spontaneous origin. Spontaneous incoördinate movements appeared sometimes before, sometimes after the light reflex, but afterward only when it returned relatively early. Coördinate movements followed quickly in dogs subjected to short periods of anemia, so that perhaps the incoordinate did not appear, but the resumption of muscular activity was sudden, the dog starting up as though suddenly roused from sleep. Succeeding the coördinate movements there came what may be classed as purposeful movements, involving all the muscles of locomotion and expressed in such actions as attempts to turn over, to rise, or to crawl forward. Usually after the appearance of coördinate movements, less frequently about the same time, visual and auditory reactions were obtained. The auditory was always the more definite and usually returned first. It is the combination of the return of the special senses with purposeful muscular movements, altogether or in part, that is termed consciousness, though it is usually very dim and uncertain. In many of the dogs that succumbed, after some hours there was more than a mere reflex revival, there being some manifestation of the higher faculties in addition.

SUMMARY. To determine the limits of recovery after a total anemia of the central nervous system, 20 dogs were killed by chloroform and resuscitated after varying times from three to fourteen minutes. Under five minutes the recovery of function was rapid and strikingly free from the after-effects which characterized longer periods. Of seven animals, resuscitated between the periods of five and six and one-half minutes, only one died, apparently as a direct result of the anemia; but of 12 resuscitated between the periods of seven and eight and one-half minutes, only one, after seven and one-half minutes, recovered. The remaining dogs all died.

Histological examination both of presumptive recoveries and fatal cases was made by ordinary methods and those of Nissl and Marchi. The neurocytes of the fatal cases uniformly presented the greatest changes, not merely chromolytic but here and there definitely indicative of cell death. Marchi's method further supported these findings by proving the existence of fiber degeneration. Finally, showing the narrowness of the escape, the best result in recovery, seven and one-half minutes in time, which at the end of four weeks had apparently entirely returned to a normal state, by the Marchi method had a degeneration of a number of fibers localized in the pyramidal fasciculi which were traced from the cord to the cortex, and in Flechsig's fasciculus, as well as a more sparsely scattered degeneration of both ascending and descending fibers elsewhere.

In human resuscitation the technique is as follows: The patient in the prone posture is subjected at once to rapid rhythmic pressure upon the chest, with one hand on each side of the sternum. This pressure produces artificial respiration and a moderate artificial circulation. A cannula is inserted toward the heart into an artery. Normal saline, Ringer's, or Locke's solution, or, in their absence, sterile water, or, in extremity, even tap water, is infused by means of a funnel and rubber tubing. But as soon as the flow has begun the rubber tubing near the cannula is pierced with the needle of a hypodermic syringe loaded with 1 to 1000 adrenalin chloride and 15 to 30 minims is at once injected. Repeat the injection in a minute if needed. Synchronously with the injection of the adrenalin the rhythmic pressure upon the thorax is brought to a maximum. The resulting artificial circulation distributes the adrenalin that spreads its stimulating contact with the arteries, bringing a wave of powerful contractions and producing a rising arterial, hence coronary pressure. When the coronary pressure rises to, say, 40 mm. or more, the heart is likely to spring into action. The first result of such action is to spread still further the blood pressure-raising adrenalin, causing a further and vigorous rise in blood pressure, possibly even doubling the normal. The excessively high pressure is most favorable to the resuscitation of tissue, especially of the central nervous system (Stewart). Just as soon as the heart beat is established the cannula should be withdrawn, first, because it is no longer needed, and second, because the rising blood pressure will drive a torrent of blood into the tube and funnel. Unless there has been hemorrhage, the only object in the use of saline infusion is to serve as a means of introducing the adrenalin into the arterial circulation toward the heart. Bandaging the extremities and abdomen tightly over masses of cotton is very useful.

From a personal experience in attempts at resuscitation of the human being, I have been impressed by two main facts: (1) The human heart seems to respond even more readily than the heart of a dog; (2) the possibility of drilling an operative staff so that the technique may be begun within two minutes.

Prior to our knowledge as to the remarkably rapid degeneration of the central nervous system I attempted a variety of cases, including drowning, electrocutions from live wire accidents, and traumatisms. I usually succeeded in establishing a heart beat, sometimes a fairly good respiratory rhythm. In one case the patient lived some days, but did not regain consciousness. Only one recovered. The most interesting case was that done by my staff at Lakeside Hospital. In the course of a neck dissection the patient suddenly collapsed. The heart, the vasomotor, the cardiac, and the respiratory centres, after approximately nine minutes of total anemia, were resuscitated. The patient died in twenty-four hours. Consciousness did not return; the pupils remained dilated, the lids half closed; there was complete muscular relaxation; the special senses were all paralyzed; all reflexes were lost; he was in appearance and in fact a decerebrate.

This method of resuscitation may be quickly executed. It involves but a slight operation. It does not in itself complicate the patient's chance for ultimate recovery.

Centripetal arterial infusion with adrenalin combined with rhythmic pressure on the chest seems to be a more effective method than that by the use of stimulants, nitroglycerin, intravenous infusions, electricity, needling the heart, and even of direct cardiac massage. We must admire the courage and the boldness of the surgeons who have attempted resuscitation by direct cardiac massage. According to our present views, in many of these cases the brain was dead before the cardiac massage was even begun. There are degrees of anemia, hence there are degrees of resuscitation.

In hemorrhage and in shock the blood pressure may be so low as to cause temporary damage to the central nervous system. An analogous degeneration is seen in pernicious anemia. Bearing in mind its grave consequences, anemia should not, if possible, be permitted beyond a certain degree. Should other measures fail, the direct transfusion of blood is, in uncomplicated cases, virtually a specific remedy. Not only the various tissues and organs of the body but the various component histological elements have an unequal endurance of anemia. Bone, connective tissue, muscle, skin, abdominal and thoracic viscera, special glands, and the heart and bloodvessels endure anemia many times longer than the central nervous system. The medulla endures anemia much better than the cerebrum. The higher the function the greater is the susceptibility to anemia. Histological changes are definite. Resuscitation to be effective must be timely. Timely resuscitation can only be done by having ever in readiness the materials needed for the technique.

THE INFLUENCE OF EMOTIONAL STATES ON THE FUNCTIONS OF THE ALIMENTARY CANAL.[1]

By W. B. Cannon, M.D.,
GEORGE HIGGINSON PROFESSOR OF PHYSIOLOGY IN THE HARVARD MEDICAL SCHOOL, BOSTON.

The contraction of bloodvessels with resulting pallor, the pouring out of "cold sweat," the stopping of salivary secretion so that the "tongue cleaves to the roof of the mouth," the dilatation of the pupils, the erection of the hairs, the rapid beating of the heart, the hurried breathing, the trembling and twitching of facial muscles, especially those of the lips—all these bodily changes are well recognized attendants of major emotional disturbances, such as fear, horror, and deep disgust. It is well known also that these changes occur chiefly in structures supplied with smooth muscle and innervated through the sympathetic nervous system. But these bodily emotional alterations, commonly noted, are mainly superficial and readily observable. Even the increased rapidity of the heart beat manifests itself in the periphery. There are, however, other viscera, supplied with smooth muscle and innervated by sympathetic fibers, which are hidden deeply in the body and which do not reveal so obviously as do the structures already mentioned the disturbances of function accompanying affective states. Special methods must be used to determine whether these organs also are included in the complex of an emotional agitation.

The bladder, for example, as Mosso has shown, is extraordinarily sensitive to mental states involving interest and attention.[2] That the tone of the bladder is much increased during excitement might be inferred from the common experience of soldiers before moving

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[1] In the use of the term "emotion" in this paper the meaning is not restricted to violent affective states, but includes "feelings" and other affective experiences. The term is also used in the popular manner, as if the "feeling" preceded the bodily change.

[2] Decennial Celebration, Clark University, 1899, p. 396.